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FILE COVERS 1907 - 8 Mar 2009 VOL 150 ISS 11
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=> s (androgen receptor) and (codon 741)
37429 ANDROGEN
29411 ANDROGENS
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794559 RECEPTOR
732160 RECEPTORS
951554 RECEPTOR
(RECEPTOR OR RECEPTORS)
11973 ANDROGEN RECEPTOR
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42806 CODON
19224 CODONS
52021 CODON
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L1 1 (ANDROGEN RECEPTOR) AND (CODON 741)

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L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2003:39630 CAPLUS
DN 139:17235
TI Novel Mutations of Androgen Receptor: A Possible
Mechanism of Bicalutamide Withdrawal Syndrome
AU Hara, Takahito; Miyazaki, Jun-ichi; Araki, Hideo; Yamaoka, Masuo; Kanzaki,
Naoyuki; Kusaka, Masami; Miyamoto, Masaomi

CS Pharmaceutical Research Laboratories I, Takeda Chemical Industries, Ltd.,
Osaka, 532-8686, Japan

SO Cancer Research (2003), 63(1), 149-153

CODEN: CNREA8; ISSN: 0008-5472

PB American Association for Cancer Research

DT Journal

LA English

AB Most prostate cancers (PCs) become resistant to combined androgen blockade therapy with surgical or medical castration and antiandrogens after several years. Some of these refractory PCs regress after discontinuation of antiandrogen administration [antiandrogen withdrawal syndrome (AWS)]. Although the mol. mechanisms of the AWS are not fully understood because of the lack of suitable exptl. models, one hypothesis of the mechanism is mutation of androgen receptor (AR). However, bicalutamide, which has become the most prevalent pure antiandrogen, does not work as an agonist for any mutant AR detected thus far in PC. To elucidate the mechanisms of the AWS, we established and characterized novel LNCaP cell sublines, LNCaP-cxDs, which were generated in vitro by culturing androgen-dependent LNCaP-FGC human PC cells in androgen-depleted medium with bicalutamide to mimic the combined androgen blockade therapy. LNCaP-FGC cells did not grow at first, but they started to grow after 6-13 wk of culture. Bicalutamide stimulated LNCaP-cxD cell growth and increased prostate-specific antigen secretion from LNCaP-cxD cells both in vitro and in vivo. Sequencing of AR transcripts revealed that the AR in LNCaP-cxD cells harbors a novel mutation in codon 741, TGG (tryptophan) to TGT (cysteine; W741C), or in codon 741, TGG to TTG (leucine; W741L), in the ligand-binding domain. Transactivation assays showed that bicalutamide worked as an agonist for both W741C and W741L mutant ARs. Importantly, another antiandrogen, hydroxyflutamide, worked as an antagonist for these mutant ARs. In summary, we demonstrate for the first time that within only 6-13 wk of in vitro exposure to bicalutamide, LNCaP-FGC cells, whose growth had initially been suppressed, came to use bicalutamide as an AR agonist via W741 AR mutation to survive. Our data strongly support the hypothesis that AR mutation is one possible mechanism of the AWS and suggest that flutamide might be effective as a second-line therapy for refractory PC previously treated with bicalutamide.

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